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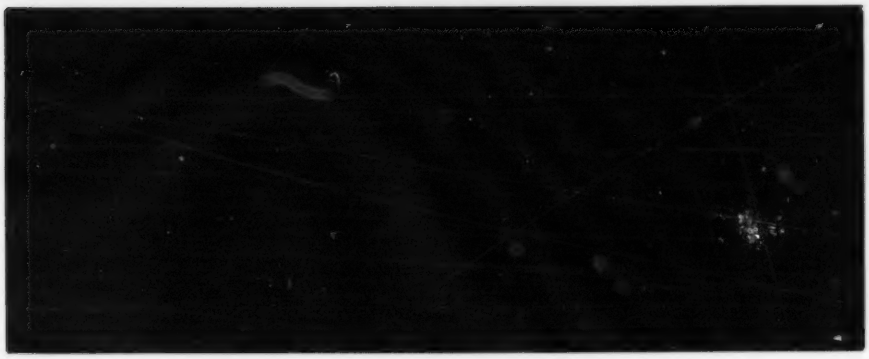

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Final Report

2.4-36

The American Elk and Meningeal Worm: Do Elk Survive It? Can They Transmit It? Can We Detect It?

Conducted by William Samuel

Funded by the Agriculture Development Fund

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EXECUTIVE SUMMARY

Project Title: THE AMERICAN ELK AND MENINGEAL WORM: DO ELK SURVIVE IT? CAN THEY TRANSMIT IT? CAN WE DETECT IT?

Principal Investigator: William M. Samuel
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This project was funded by the Canada-Saskatchewan Subsidiary Agreement for Agriculture under the Economic and Regional Development Agreement (ERDA)

ABSTRACT: Ten captive elk calves were infected experimentally with the parasitic nematode, meningeal worm (Parelaphostrongylus tenuis), to determine if: 1) elk can survive infection; 2) the worm can development normally in elk; and 3) whether analysis of feces is a viable way to detect infection. The research was conducted to assist Saskatchewan Agriculture officials in determining the potential for meningeal worm to be imported into Saskatchewan by the game growing industry. Each of five elk given 300 larvae died of neurologic disease. Many adult worms were recovered at necropsy and a few larvae were recovered from feces of the longest surviving elk (the animal was seriously ill when larvae were discovered). Each of five elk given 14 larvae survived with no clinical signs, no larvae were shed in feces, and only a total of five adult worms were recovered at necropsy. In summary, elk are susceptible to neurologic disease caused by meningeal worm but some survive infection; the worm develops normally in elk but some worms penetrate nervous tissue resulting in neurologic illness and, often, death; analysis of feces is only a good method of detection in lightly infected elk when many sequential analyses are run on one animal.

SUMMARY: Meningeal worm (scientific name, Parelaphostrongylus tenuis) is a normal, very prevalent, and non-pathogenic parasite of white-tailed deer of eastern North America. In Canada, P. tenuis has been reported from deer as far west as western Manitoba; it is **not** known from any animals in Saskatchewan, Alberta, or British Columbia. It produces no ill effects in whitetails, but does produce an often fatal neurologic disease in many other species of mammals including moose, elk, woodland caribou, red deer, reindeer, fallow deer, black-tailed deer, mule deer, domestic goats and sheep, and a variety exotic/zoo ungulates such as llamas.

Ten captive elk were experimentally infected with meningeal (or brain) worm at the University of Alberta during the winter-spring of 1989. The major objectives of the study were:

1) to determine if elk are so susceptible to neurologic disease caused by the parasite that all infected animals die, and;
2) to determine if meningeal worm can develop normally in elk to the point where adult worms shed juvenile parasites (called larvae) in elk feces, thus completing the cycle as it occurs in white-tailed deer.

The research was conducted in order to assist Saskatchewan government officials in determining the potential of an accidental importation of meningeal worm from eastern North America with elk by game ranchers. A subjective non-quantitative evaluation of the Baermann technique for examination of feces was added to determine if the technique is useful as a routine quarantine testing procedure for elk being brought into Saskatchewan from eastern North America. Initial efforts into development of a diagnostic serologic test were attempted.

Each of 5 elk received 15 larvae (termed the low-dose group) and each of 5 received 300 larvae (termed the high-dose group). Two other elk were used as uninfected controls. Two white-tailed deer were exposed to the same source of dose-larvae as given the elk; one (a fawn) received 15 larvae and the other (an old adult that had previously been infected several years previously with a close relative of meningeal worm), 200. Unfortunately, no other deer were available.

The young control deer began producing larvae 86 days after exposure. It yielded 14 adult worms at necropsy. Both these results suggest that the white-tailed deer is a very normal host for meningeal worm.

None of the control or low-dose elk became ill and none ever shed larvae during the 150 days that feces were checked following exposure to the parasite. A total of 5 adult worms were recovered from 2 of these elk.

In contrast, all five high-dose elk died of neurologic disease at 60,71,94,102, and 111 days after exposure, respectively. Of the three elk that lived long enough for

larvae to have been shed in the feces, only one shed, and that only a few larvae.

In summary, we have shown that although elk are sensitive to meningeal worm and some die of a worm-induced neurologic disease, lightly infected elk survive though they shed no larvae. Infected elk differ from white-tailed deer in that some meningeal worms in elk penetrate nervous tissues causing disease. That a potential problem exists regarding transmission (and, hence, introduction of this parasite into Saskatchewan) was revealed by the longest surviving high-dose elk. This elk shed a total of 31 larvae between day 105 and 111 post-exposure. It was seriously ill with neurologic disease during this time.

The Baermann Technique, as used routinely to survey feces of elk for meningeal worm; that is, a one-time or one-per-day 20 gram sample of fresh feces from each elk is marginal at best in detecting very low levels of larvae. During the time (i.e., 105 to 111 days of the infection) the one elk shed larvae, 28 fecal samples were run. One (53 grams of feces) yielded 23 larvae and six (50,52,56,42,50, and 68 grams of feces) yielded a total of 8 larvae. Many (13) of the remaining 21 negative fecal samples weighed over 40 grams. The point is that many fecal samples must be run sequentially to detect very light infections.

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The young control deer began producing larvae 86 days after exposure. It yielded 14 adult worms at necropsy. Both these results suggest that the white-tailed deer is a very normal host for meningeal worm.

The older control whitetail, which had had an earlier infection with a close relative of meningeal worm, never shed larvae and yielded only one adult worm at necropsy 165 after exposure. It developed a strong immune response to the infection, showing clinical signs of neurologic disease early in the infection and yielding only one adult meningeal worm. The clinical signs continued in varying degrees until

the animal was killed. The earlier experimentally infection with muscle worms (classified in the same genus as meningeal worm) probably provided some resistance to the meningeal worm infection.

None of the control or low-dose elk became ill and none ever shed larvae during the 150 days that feces were checked following exposure to the parasite. A total of 5 adult worms were recovered from 2 of these elk.

In contrast, all five high-dose elk died of neurologic disease at 60,71,94,102, and 111 days after exposure, respectively. Of the three elk that lived long enough for larvae to have been shed in the feces, only one shed, and that only a few larvae.

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BACKGROUND ON MENINGEAL WORM, PARELAPHOSTRONGYLUS TENUIS:

The disease: parelaphostrongylosis, cerebrospinal nematodiasis, moose sickness

Scientific name of parasite and normal host: Parelaphostrongylus tenuis; Odocoileus virginianus.

Common name of parasite and normal host: meningeal worm (often called brainworm); white-tailed deer.

Known geographic distribution: Eastern and Central Canada including Nova Scotia, New Brunswick, southern Quebec, Ontario, and Manitoba. Eastern and Central United States including Maine, New Hampshire, Connecticut, New York, Pennsylvania, Maryland, New Jersey, West Virginia, Virginia, North Carolina, South Carolina, Georgia, Florida, Alabama, Tennessee, Kentucky, Michigan, Indiana, Wisconsin, Minnesota, Iowa, Missouri, Mississippi, Louisiana, Arkansas, Missouri, Texas, and Oklahoma.

Clinical signs of the disease: the white-tailed deer is the normal host for this parasite and it displays few clinical signs. Anderson (1965b) and Prestwood (1970) describe a temporary lameness of the forelimb, and circling and rapid oscillation of the eyeballs, respectively, in white-tailed deer with experimental infections.

In moose and the many other hosts debilitated by infection, the disease is expressed by locomotor incoordination, lameness, stiffness,

listlessness, weakness of the hind quarters, circling, abnormal positions of the head and neck, blindness, and paralysis.

Major pathologic changes: Extensive lesions of the central nervous system including focal hemorrhaging, neuron degeneration, and yellowish accumlations streaked with blood near worms. Worms are found free in the cranial cavity or on the spinal cord, or partially embedded in nervous tissue. For more detail see Anderson and Prestwood (1981), Anderson and Strelive (1967), Gilbert (1973), and many others.

Method of diagnosis: The method of choice is recovery and identification of the adult worm; this of course means that the host must be sacrificed. Recovery of the first-stage larvae in feces using a modified Baermann Technique (see Samuel and Gray, 1982 for a description of this technique) is a step in the right direction, but it must be realized that several other parasitic nematodes of North America shed similar cork-screw, spiny-tailed larvae. These include Muellerius capillarus (a lungworm of domestic goat and sheep, but also known from bighorn sheep; Pybus and Shave, 1984), Varelaphostrongylus alpenae (a lungworm of white-tailed deer; Gray et al., 1985), Parelaphostrongylus andersoni (a muscle worm of white-tails [see Anderson and Prestwood, 1981] and caribou, Lankester and Hauta, 1989), Parelaphostrongylus odocoilei (a muscleworm of mule deer, see Platt and Samuel,1978b), and Elaphostrongylus cervi (a nematode of the muscles and nervous tissue of caribou, see Lankester and Northcott, 1979).

Lengths of some of these larvae differ (Gray et al., 1985) with V. alpenae and M. capillaris being short and E. cervi, long (Gray et al., 1985), but there is much overlap.

Ballantyne and Samuel(1984) differentiated the infective larvae (i.e., the third-stage larvae found in terrestrial snails and slugs) of the three species of Parelaphostrongylus using morphology of the tail. Some (Upshall et al. 1986) have found it useful, others (Lankester and Hauta, 1989) not.

Due to the difficulty in diagnosing the infection in live animals, several authors simply recommend that known host species not be translocated from areas where the parasite occurs to new areas.

An important but as yet undeveloped detection method is a serological (blood) test , the Enzyme-Linked Immunosorbent Assay (ELISA), for detection of meningeal worm infection in elk. If developed

this test will be fast, reliable and reporducible and will eliminate the need for examination for parasites in the feces.

Method of treatment: Currently there are no drugs known to be 100% effective against meningeal worm or its close relatives, but little research has been done (Samuel and Gray, 1988). Apparently, clinically ill llamas have been treated successfully.

Other species infected and affected by the same organism: Moose, elk, caribou, reindeer, mule deer, black-tailed deer, mule deer-whitetail hybrids, fallow deer, red deer, red deer-elk hybrids, domestic sheep, domestic goat, llamas, guinea pigs, and several bovid and cervid species in zoos.

Prevalence of the parasite: Prevalence of meningeal worm in deer ranges from <1% to more than 85% in various parts of North America. In Canada, prevalence figures for adult worms in the cranial cavities of deer are: 10% (Manitoba), 41-61% (Ontario), 30% (Quebec), 60% (New Brunswick), and 51% (Nova Scotia).

Prevalence of meningeal worm in other hosts is largely unknown although a recent survey in Nova Scotia (Thomas and Dodds, 1988) report it in 6.5% of the moose there. Woolf et al.(1977) report it in from 27 to 64% of a wapiti population in Pennsylvania.

Although prevalence data are hard to come by, there are some interesting semi-quantitative papers (see Saunders,1973). Also, there are many case reports of neurologic disease in moose, some for elk, and up to five reports of disease in other species listed above. Subjective assessments of the importance of the disease abound in the literature. It is probable that serious discussion of this parasite is part of every wild ungulate translocation in eastern North America, because of the documented failures of several reintroduction programs.

Importance to domestic species: Based on the relatively few case reports of disease in sheep and goat from Michigan, New York, New Hampshire, Connecticut, West Virginia, Ohio, and Texas, this disease is not that important for domestic livestock. However,"the disease may be more common in livestock than is indicated by the literature" (Anderson and Prestwood, 1981). Cattle must have some degree of resistance, at least based on the fact that no cases of disease have been reported. Llamas are very susceptible to disease and death. Deaths have been reported from Michigan, Virginia, Wisconsin, Texas, and Ohio.

Public health implications: None known

BACKGROUND TO THE STUDY:

The inadvertent introduction of parasites and diseases with deliberate movement of animals to new geographic locations is well documented. Some of the better known examples of transcontinental introductions from a wild or domestic animal source include: rinderpest, which didn't occur in much of Africa until European settlers introduced cattle; the hydatid tapeworm, Echinococcus granulosus, introduced into various parts ofthe world with introductions of various species of deer, the liver fluke Fascioloides magna introduced into Europe with American elk, and whirling disease, a protozoan introduced from Europe into North America with brown trout in the late 1950's, eradicated from Europe, then reintroduced there from North America.

There are less famous examples such as the transfer of a chewing louse from the introduced fallow deer to the Columbian black-tailed deer in California, the introduction of a tapeworm pathogenic for cyprinid fish introduced into Europe, Russia, and the United States in grass carp from China, or the introduction of an avian eye fluke with a zoo animal and its snail intermediate host by suppliers for aquaria into Texas. Once introduced, a parasite or disease agent can become established when only a few general/basic factors are met. These include the presence of susceptible hosts and adequate means to spread the parasitic/disease agent, which means the proper external environmental conditions for survival of the parasite are met.

Are there current circumstances by which parasites/diseases could be translocated with its host? In western Canada, two plausible avenues exist. One is a program of trapping and transplanting wild species from one place to another. Such operations are usually done by provincial game departments. The usual strategy is to reintroduce a wild species into areas once utilized by the wildlife species in question. A strategy seldom used is the introduction of an animal into a new area.

A second avenue involves the maintenance of wildlife in captivity for purposes of development of a game growing industry. Game growing as an agricultural alternative to more traditional practices is thriving in Western Canada.

In North America, meningeal worm has been found in whitetails as far west as the Manitoba-Saskatchewan and Minnesota-North Dakota borders in the north to eastern Oklahoma in the south.

Meningeal worm is either not spreading westward in Canada naturally via contiguous whitetail populations residing throughout southern and central Saskatchewan and Alberta or is doing so very slowly. A natural

ecologic barrier of unknown width might exist on the Manitoba-Saskatchewan border. However, because white-tailed deer and the intermediate hosts (a slug and various land snails) are common (actually, quite abundant) in parts of Saskatchewan and Alberta, P. tenuis could become established if introduced with a translocated host such as elk.

Samuel (1987) has reviewed the potential negative impact of importing elk into western Canada from areas where P. tenuis occurs. Concerns raised are only valid if meningeal worm can successfully complete its life cycle in elk; i.e., spined first-stage larvae of meningeal worm are passed in elk feces. Three papers suggest that this is possible (Karns 1966, Anderson et al.1966, Pybus et al., 1989); however, the data in all three papers are only suggestive not conclusive.

It should be relatively easy to keep the natural host, white-tailed deer from eastern North America out of Saskatchewan and Alberta because they are not prized animals for game growing and sufficient local stock exists. Elk, however, are the most prized animal for game growing in the West and we should find out how suitable are these hosts for development (and transmission) of P. tenuis.

OBJECTIVES:

1) to determine if elk are so susceptible to neurologic disease caused by meningeal worm that all infected animals die;
2) to determine if meningeal worm can develop normally in elk to the point where adult worms shed juvenile parasites (called larvae) in elk feces, thus completing the cycle (as occurs in white-tailed deer), and;
3) to initiate development of a diagnostic serologic test for meningeal worm in elk (an objective we added to our original proposal knowing that there was essentially no funding for this aspect, but also knowing that local expertise and interest existed at the University of Alberta).

EXPERIMENTAL METHODS:

1). We reviewed current stringent regulations at the Ellerslie Research Station, University of Alberta, to guarantee that meningeal worm could not be introduced into local, free-ranging, wild ruminants (a program has been in place since 1975 and there has never been a problem of any kind).

2). In November, 1988, we acquired a pure infection of meningeal worm larvae from feces of whitetails of the Rachelwood Wildlife Preserve in western Pennsylvania, a site where meningeal worm is prevalent in white-tailed deer and neurologic disease has been reported commonly in elk.

3). We exposed our laboratory culture of terrestrial gastropods, Triodopsis multilineata, (a known good intermediate host of meningeal worm) to the larvae from the deer feces.

4). We acquired two fawn white-tailed deer through the AB Fish and Wildlife Division to use as controls for the infection in December, 1988. One of the fawns received larvae from a source in Michigan that proved to be contaminated with another parasite that produces a dorsal-spined larvae, so that fawn was dropped from the experiment as was the Michigan source of larvae. I was then forced to use an old whitetail doe in my possession as the second infected control animal. Another very old whitetail doe of mine was used as an uninfected control. Since she never shed larvae, she was not killed.

5). We acquired elk calves from Bob Hudson (n=5), University of Alberta, and seven from Alex Hawley of the Alberta Environmental Centre, Vegreville, Alberta, in early December, 1988.

6). We infected 1 fawn with larvae from a Michigan source, but that did not work out (see "4)" above), so no data for that animal are presented here. We infected the second fawn with 15 larvae of meningeal worm and the old doe with 200 larvae from a pure, good Pennsylvania (see "2)" above)

7). On January 4 and 5, 1989 we infected 5 elk calves each with 200 (high dose) and 15 (low dose) of infective stage meningeal worm larvae that we digested from our snails (Table 1). The larvae were administered to the tranquilized elk by stomach tube. We kept two remaining elk as uninfected controls.

Table 1. Experimental design.

Species	Number of elk in the exp't.	Sex	Dose
Elk	3	Female	300
	2	Male	300
	4	Male	15
	1	Female	15
White-tailed Deer			
fawn	1	Male	15
old adult	1	Female	200

8). We monitored feces of each animal for passage of meningeal larvae beginning 7 days before exposure of animals to the parasite. A modified Baermann Technique was run (2,230 fecal samples were run). Twenty grams of fresh feces were collected in individual plastic bags and stored refrigerated or frozen until examined for spine-tailed larvae. These feces were weighed and put on cheesecloth in a glass water-filled funnel. They were allowed to stand overnight, then in the morning, 100 ml. of water was drawn off and examined for larvae.

Collections were done weekly until 40 days after infection, then daily until the end of the experiment. Deer feces were checked daily for larvae while elk feces were checked every three days. If larvae were seen in elk feces, the previous two daily samples that had been collected but not examined, were examined.

9). Serum was collected from all animals at regular intervals (twice per month) for use in development of the serologic ELISA test. Two main approaches will be used for the development of the tests:

1).Detection of parasite-specific antibodies in the serum of experimentally infected elk. This test will tell us whether elk were previously exposed to the parasite;

and 2). detection of parasite antigens (molecules unique to the parasite) in the serum and body secretions (such as saliva and serum) of experimentally infected elk. This test will tell us whether elk is currently infected with the parasite.

The first approach has been initiated; if there is no cross reactivity with other nematodes, a test could be developed in a year. If there is cross reactivity between meningeal worm and other nematodes such as the cattle lungworm, Dictyocaulus viviparus, then critical specific antigens must be identified. This will add 2 years to the development of the test. The second will require 2 to 3 years to complete.

10).All animals were monitored closely for signs of neurologic disease. Details of altered behavior were recorded. Animals showing clinical signs were video-taped. When it became obvious that an animal was in severe condition and would die soon, it was killed with sodium pentabarbital (Euthanyl) administered intraveneously. A detailed necropsy was done on each animal. Representative tissues for histology were collected from all animals; Alberta Agriculture prepared tissues for examination. Margo Pybus, with imput from Detlef Onderka of Alberta Agriculture, did the histo-

pathology. Margo identified the parasites recovered.

11). All remaining animals were killed and necropsied by 165 days after infection.

Detailed pathology and details of worm locations will not be presented here due to the extensive and technical nature of the data and the fact that those results are more academic in interest and are not really all that important to the contract-related part of the study. These data will be published in a scientific journal.

RESULTS:

A total of 12 elk and 2 white-tailed deer were necropsied during this project. I and 3 fantastic dedicated research associates and technicians worked many long hours to make this project worthwhile. Chris Wilke examined 2,230 samples of feces using the Baermann Technique; 1,631 of these were on elk.

High-dose elk - All five elk receiving 300 meningeal worm larvae died of neurologic disease between 60 and 111 days after exposure to the parasite larvae (Table 2). Adult

Table 2. Neurologic disease and worm recovery information in high-dose(300 worms) elk.

Elk	Sex/Age	No. Days with Clinical Signs Prior To Death	Days After Exposure That Elk Died	No. worms Recovered
E115	F 8mo.	2	60	53
E017	M 8mo.	6	71	57
E114	F 8mo.	23	94	34
E005	M 8mo.	3	102	28
E117*	F 8mo.	19	111	28

*Only animal that shed larvae of meningeal worm in feces.

worms consistent with morphologic descriptions of Parelaphostrongylus tenuis were recovered from the brain and spinal cord of each elk (Table 2). A total of 200 worms were collected from the five elk. Worms were distributed along the entire length of the central nervous system from the brain to the posterior part of the spinal cord. Of the 200 worms recovered, 35% were from the brain, and 18, 16, 26, and 3 % were from the cervical, throacic, lumbar, and sacro-caudal regions, respectively. An additional 2.5% were recovered in sites outside the central nervous system.

Most worms in the cranial cavity were in locations considered normal (examples: in the subdural space - 33 of 70 worms; or in the leptomeninges - 25 of 70 worms). Only 5

of 70 worms in the cranial cavity were embedded deeply in brain tissue, but these five were distributed in the two elk that died last.

Of the 128 worms found along the spinal cord, most were in normal locations (examples: in the subdural space - 79 of 128 worms; or in the leptomeninges - 7 of 128 worms). Twenty were in epidural regions mainly in association with spinal ganglia while 18 were embedded in spinal cord gray or white matter.

Elk were clinically ill from 2 to 23 days (Table 2). Clinical signs usually developed as follows:

1) elk became quieter than normal (2 to 13 days in duration in 3 elk);
2) elk became stiff in hind quarters (1 to 7 days in duration in 4 elk);
3) elk developed a mild to severe twist or kink in the neck (seen as the first clinical sign in 1 elk, and the third clinical sign in 2 elk; 3 to 9 days in duration).
4) elk became more lame in the hind quarters and often had great difficulty in rising (1 to 3 days in duration in 4 elk).
5) elk circled, often continuously (circles were usually 3 or so feet in diameter); seen in two animals for 3 and 9 days prior to death, respectively.
6) elk either could not rise or stood in deep depression with head held low and, in the end, laboured breathing (1 to 9 days in all 5 elk).

Thus, we have documented the progression of clinical disease in elk from an initial quieter-than-normal disposition and stiff gait to severe weakness and inability to rise. All elk remained alert and fed as normally as possible during progression of the disease. It was only near the development of the worst clinical signs (we did not let animals die naturally, but killed them to avoid undue suffering) that animals did not feed or drink.

Remembering that meningeal worm matures in the normal host, the white-tailed deer in 80-90 days after exposure to larvae, elk that died prior to this should not have passed larvae in their feces. Although three elk lived longer than 90 days after exposure, only one (No. E-117) shed larvae of meningeal worm in its feces (Table 2). That elk first shed 23 larvae in a 53 gram sample of feces (that is: 0.43 larvae per gram of feces) 105 days after exposure to the parasite. Between days 106 and 111, when this elk was killed with severe disease, all feces were collected and examined. A total of 27 fecal samples were examined using the Baermann Technique. Six were positive for larvae, but only a total of 8 larvae were found. All 6 positive fecals were collected on day 109.

Because larvae leaving the host body via the feces must first pass through the lungs, all lungs of all exposed

animals were Baermannized. Minced lung tissue from E-117 placed in 34 Baermann funnels yielded only 1 larvae.

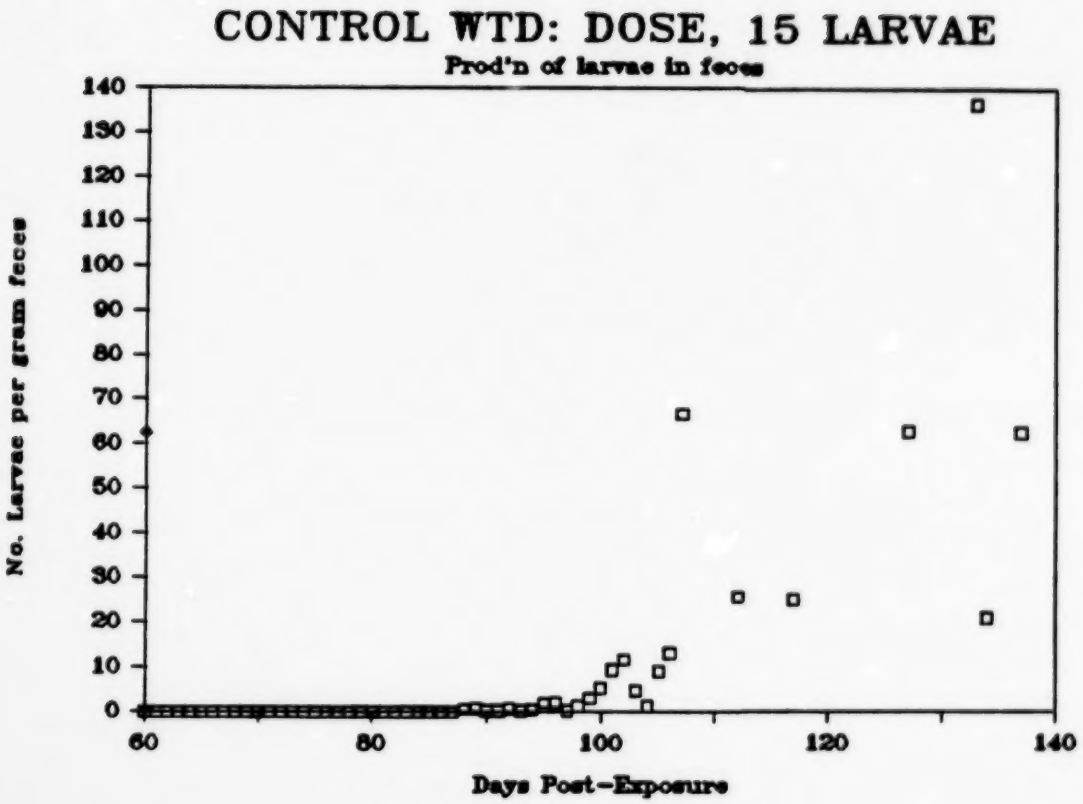
The lungs of the second-longest surviving elk, E-005 (see Table 2) yielded seven larvae of meningeal worm. All other fecals and lungs were negative.

Low-dose elk - None of the control elk or the five elk receiving 15 larvae of meningeal worm exhibited clinical neurological or behavioural signs. None shed larvae. A total of five adult worms were recovered at necropsy (Table 3) including two adult females on the brain and 1 adult male in the cervical region of the spinal cord of E018 and one adult female on the brain and one unsexed adult in the cervical region of the spinal cord of E111. No worms or evidence of infection was seen in central nervous system tissue.

Table 3. Recovery of adult meningeal worm from elk given 15 larvae.

Elk Number	Sex/Age	Days After Exposure When Elk Were Killed	Worm Recovery
INFECTED ELK:			
E018	M 8mo.	120	3
E116	M 8mo.	143	0
E003	M 8mo.	147	0
E113	M 8mo.	154	0
E111	F 8mo.	159	2
CONTROL ELK:			
E020	M 8mo.	124	0
E112	M 8mo.	161	0

Control deer - The white-tailed deer fawn that received 15 larvae of meningeal worm did not exhibit clinical neurological or behabioural signs. It began shedding larvae in its feces on 86 days after exposure to the worm (see next Figure).



It was killed 144 days after exposure to the parasite; a total of 14 mature worms were recovered from this animal.

The doe deer that received 300 larvae of meningeal worm exhibited clinical neurological signs in the form of partial paralysis of the hind legs (particularly the left hind leg) throughout much of the 165 days of the experiment. At times the animal was very lame, other times not. She never shed any larvae and at necropsy only one adult female worm was collected from the cranial cavity. These data suggest that this animal was resistant to meningeal worm probably because it had been infected experimentally with a muscleworm, Parelaphostrongylus andersoni, several years ago and this doe had been sensitized to the closely related meningeal worm, Parelaphostrongylus tenuis.

DISCUSSION, CONCLUSIONS AND IMPACT

The study reported here has been extended into a second phase now underway. Currently, 16 additional elk calves have been infected at doses of meningeal worm that fall between those given elk here. Thus, final decisions on this problem should probably be based upon results and conclusions from both studies. The experimental phase of the second experiment will be concluded around January 1, 1990.

Nonetheless, it is obvious that elk are sensitive to meningeal worm and are not a good host. In like manner, however, lightly infected elk (perhaps more representative of wild or captive elk in the East) can survive the infection and display no clinical signs. Beacuse all worms found were adults and many were sexually mature, it is also obvious that meningeal worm develops more or less normally in elk. The only problem is that, in some cases, some worms penetrate nervous tissue resulting in neurologic disease.

As to whether elk pose any risk of introducing meningeal worm to snail/slug intermediate hosts in Saskatchewan via shedding of larvae of the parasite in their feces, results are less clear. Eight of the 10 infected elk survived long enough (i.e., over 90 days) for the worm to shed larvae in the feces, yet only one did so, and that at a low level of larvae. In addition that animal was severely ill with disease when it began to shed larvae. Whatever, a potential introduction and transmission does exist in that the longest surviving high-dose elk shed a total of 312 larvae (in the feces examined) between day 105 and 111 of the infection.

Our conclusion? Potentially it only takes one snail/slug to become infected and later eaten by a white-tailed deer for meningeal worm to become established. How far fetched is this possibility? Who knows, but the fact remains that one infected whitetail could easily seed an area with many many larvae. And if snails are in the area (a logical conclusion in the Aspen Parklands) then the ingredients are in place. (Results of the second phase will add much more relevant data to the current data set.)

Given this, are our current diagnostic tests, (that is, the examination of feces for meningeal worm larvae) capable of detecting this parasite? We and others working on similar parasites (such as Elaphostrongylus cervi in New Zealand red deer) must state a qualified no.

For routine testing of elk for meningeal worm in Alberta and Saskatchewan, the Baermann Technique involves a one-time collection and examination of 20 grams of fresh elk feces. Chances of detecting larvae in a 20 gram fecal sample when few larvae are there are not great. For example, during the seven days that the one elk in this study shed larvae, 28 fecal samples were ren. One (53 grams of feces were placed in Baermann funnels) yielded 23 larvae and six (50,52,56,42,50, and 68 grams of feces) yielded a total of 8 larvae. Many (13) of the remaining 21 negative

fecal samples weighed over 40 grams. The point is that many fecal samples must be run (by highly qualified personnel I might add) if low levels of infection are to be detected. Sequential sampling of much feces is recommended.

CONCLUSIONS AND IMPACT:

Some will say that if the worm hasn't spread naturally into Saskatchewan with naturally spreading white-tailed deer, it never will. They would argue that there is no real or potential problem regarding introduction of this parasite into Western Canada. They reason that there must be some barrier associated with the prairies and/or aspen parklands that is preventing meningeal worm from reaching and/or establishing in Saskatchewan. This could be true, but at this point in time all we know for sure is that Saskatchewan has all the ingredients for spread of the disease if a small focus of infection became established.

Saskatchewan currently has large and widely distributed populations of both the primary host, the white-tailed deer, and (presumably) the intermediate hosts, land snails and slugs. In addition, Saskatchewan has a variety of domestic and wild ungulates most of which we know are susceptible to the disease (domestic sheep and goat, llamas, moose, elk, mule deer and pronghorn).

Potential impact on native wildlife and possibly on domestic livestock would no doubt be considerable. As an example, suppose we pick a few individuals of any of the aforementioned species and have them come down with neurologic disease; consider the political, biological, financial, and logistical consequences. Eradication of this worm, once established, would probably not be possible. And, if more than a few individuals come down with infection, multiply the hassle by some figure at your discretion. Simply put, we have no meningeal worm now so we have no short-term, minimal, long-term or major problem.

Talks given:

25 February, 1989. Importation of elk into Alberta: the meningeal worm story. M. J. Pybus. Western Canada Wildlife Disease Conference, University of Saskatchewan, Saskatoon.

13 April, 1989. Moving the zoo "OR" the potential introduction of a dangerous parasite/disease agent with its translocated host. W. M. Samuel. 40th Annual Meeting, Northwest Section of The Wildlife Society, Banff.

15-17 May, 1989. W. M. Samuel. Participant in Workshop on Wildlife Diseases and Game Growing. Saskatchewan Agriculture, Saskatoon.

15 August, 1989. The meningeal worm stroy as it relates to animal translocations in western Canada. W. M. Samuel, D. A. Welch, M. J. Pybus, and C. J. Wilke. 1989 Wildlife Disease Association 38th Annual Conference, Corvallis, Oregon.

15 August, 1989. Effect of meningeal worm, Parelaphostrongylus tenuis (Nematoda) on the American elk: an experimental study. D. A. Welch, M. J. Pybus, W. M. Samuel, and C. J. Wilke. 1989. Wildlife Disease Association 38th Annual Conference, Corvallis, Oregon.

14 September, 1989. Meningeal worm and its implications in the game growing industry in Alberta. W. M. Samuel. Departmental Seminar, Wildlife Section, Department of Forestry, West Virginia University, Morgantown, West Virginia.

6 October, 1989. The meningeal worm hazard. W. M. Samuel. Conference '89, Canadian Llama Association 2nd Annual Llama Conference, Red Deer.

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